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 NEWS  1          Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Jan 25  BLAST(R) searching in REGISTRY available in STN on the Web
 NEWS  3  Jan 29  FSTA has been reloaded and moves to weekly updates
 NEWS  4  Feb 01  DKILIT now produced by FIZ Karlsruhe and has a new update
                  frequency
 NEWS  5  Feb 19  Access via Tymnet and SprintNet Eliminated Effective 3/31/02
 NEWS  6  Mar 08  Gene Names now available in BIOSIS
 NEWS  7  Mar 22  TOXLIT no longer available
 NEWS  8  Mar 22  TRCTHERMO no longer available
 NEWS  9  Mar 28  US Provisional Priorities searched with P in CA/CAplus
                  and USPATFULL
 NEWS 10  Mar 28  LIPINSKI/CALC added for property searching in REGISTRY
 NEWS 11  Apr 02  PAPERCHEM no longer available on STN.  Use PAPERCHEM2
instead.
 NEWS 12  Apr 08  "Ask CAS" for self-help around the clock
 NEWS 13  Apr 09  BEILSTEIN: Reload and Implementation of a New Subject Area
 NEWS 14  Apr 09  ZDB will be removed from STN
 NEWS 15  Apr 19  US Patent Applications available in IFICDB, IFIPAT, and
IFIUDB
 NEWS 16  Apr 22  Records from IP.com available in CAPLUS, HCAPLUS, and
ZCAPLUS
 NEWS 17  Apr 22  BIOSIS Gene Names now available in TOXCENTER
 NEWS 18  Apr 22  Federal Research in Progress (FEDRIP) now available

 NEWS EXPRESS  February 1 CURRENT WINDOWS VERSION IS V6.0d,
               CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),
               AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
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 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)
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* * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 12:56:53 ON 15 MAY 2002

```
=> fil reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

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STRUCTURE FILE UPDATES:   13 MAY 2002  HIGHEST RN 415678-09-0
DICTIONARY FILE UPDATES:  13 MAY 2002  HIGHEST RN 415678-09-0

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> e ar-nox/cn
E1            1     AR-NITROBENZO(K)FLUORANTHENE/CN
E2            1     AR-NITROXYLENE/CN
E3            0 --> AR-NOX/CN
E4            1     AR-OCCIDOL/CN
E5            1     AR-OCTADECYLBENZENAMINE/CN
E6            1     AR-P 320/CN
E7            1     AR-P 322/CN
E8            1     AR-P 515/CN
E9            1     AR-P 525/CN
E10           1     AR-P 610.08/CN
E11           1     AR-P 661/CN
E12           1     AR-PENTABROMOSTYRENE/CN

=> e nadh oxidase/cn
E1            1     NADH KINASE/CN
E2            1     NADH NITRATE REDUCTASE (SOLANUM TUBEROSUM GENE STNR2)/CN
E3            1 --> NADH OXIDASE/CN
E4            1     NADH OXIDASE (AMPHIBACILLUS XYLANUS CLONE PNOX2)/CN
E5            1     NADH OXIDASE (AMPHIBACILLUS XYLANUS STRAIN EP01 GENE
FAP)/CN
E6            1     NADH OXIDASE (AQUIFEX AEOLICUS GENE NOX)/CN
E7            1     NADH OXIDASE (ARCHAEOGLOBUS FULGIDUS GENE AF0515)/CN
E8            1     NADH OXIDASE (ASPERGILLUS SOJAE STRAIN SU-1 GENE NADA)/CN
E9            1     NADH OXIDASE (BACILLUS HALODURANS STRAIN C-125 GENE
BH1481)/
                    CN
E10           1     NADH OXIDASE (BRACHYSPIRA AALBORGI STRAIN ATCC-43994 GENE
NO
                    X FRAGMENT)/CN
E11           1     NADH OXIDASE (BRACHYSPIRA HYODYSENTERIAE STRAIN B169 GENE
NO
                    X FRAGMENT)/CN
E12           1     NADH OXIDASE (BRACHYSPIRA HYODYSENTERIAE STRAIN B78 GENE
NOX
```

```
                    FRAGMENT)/CN

=> s e3
L1            1 "NADH OXIDASE"/CN

=> d

L1   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2002 ACS
RN   9032-21-7  REGISTRY
CN   Oxidase, reduced nicotinamide adenine dinucleotide (9CI)  (CA INDEX NAME)
OTHER NAMES:
CN   Dihydrocodehydrogenase I oxidase
CN   Diphosphopyridine nucleotide oxidase
CN   DPNH oxidase
CN   NAD oxidase
CN   NADH oxidase
CN   NADH-oxygen reductase
CN   NADH2 oxidase
CN   Reduced nicotinamide adenine dinucleotide oxidase
MF   Unspecified
CI   MAN
LC   STN Files:   AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS,
       CHEMCATS, CIN, EMBASE, PROMT, TOXCENTER, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
             1466 REFERENCES IN FILE CA (1967 TO DATE)
               14 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
             1469 REFERENCES IN FILE CAPLUS (1967 TO DATE)


=> fil caplus uspatfull biosis embase medline
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       5.96       6.17

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FILE 'MEDLINE' ENTERED AT 12:57:44 ON 15 MAY 2002

=> morre dorothy/au
MORRE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> e morre dorothy/au
E1            1     MORRE D L/AU
E2          336     MORRE D M/AU
```

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E3            1 --> MORRE DOROTHY/AU
E4          187     MORRE DOROTHY M/AU
E5            1     MORRE DOROTHY MARIE/AU
E6            9     MORRE E/AU
E7            1     MORRE E E/AU
E8            1     MORRE ECKHART/AU
E9            1     MORRE F A/AU
E10           1     MORRE F D/AU
E11           1     MORRE F L/AU
E12           1     MORRE G/AU

=> s e3 or er or e5 or e2
L2       105112 "MORRE DOROTHY"/AU OR ER OR "MORRE DOROTHY MARIE"/AU OR "MORRE
                D M"/AU

=> s morre d/au
L3            7 MORRE D/AU

=> e morre d/au
E1            1     MORRE BOOKER T/AU
E2            1     MORRE CHRIS C D/AU
E3            7 --> MORRE D/AU
E4            1     MORRE D E/AU
E5         1213     MORRE D J/AU
E6            1     MORRE D J MORRE AND D M/AU
E7          539     MORRE D JAMES/AU
E8            1     MORRE D L/AU
E9          336     MORRE D M/AU
E10           1     MORRE DOROTHY/AU
E11         187     MORRE DOROTHY M/AU
E12           1     MORRE DOROTHY MARIE/AU

=> s e3 or e5 or e6 or e7
L4         1756 "MORRE D"/AU OR "MORRE D J"/AU OR "MORRE D J MORRE AND D M"/AU
                OR "MORRE D JAMES"/AU

=> s l4 and l2
L5          304 L4 AND L2

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6          186 DUP REM L5 (118 DUPLICATES REMOVED)

=> s l6 and ar-nox
L7            0 L6 AND AR-NOX

=> s l6 and nadh oxidase
L8           59 L6 AND NADH OXIDASE

=> s screen or screening
L9       986566 SCREEN OR SCREENING

=> s l8 and l9
L10           1 L8 AND L9

=> d ibib abs

L10  ANSWER 1 OF 1  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER:   2002136537  EMBASE
TITLE:              Monoclonal antibody to a cancer-specific and
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drug-responsive hydroquinone (**NADH**) **oxidase** from the sera of cancer patients.

AUTHOR: Cho N.; Chueh P.-J.; Kim C.; Caldwell S.; **Morre D.M.**; **Morre D.J.**

CORPORATE SOURCE: D.J. Morre, Department of Medicinal Chemistry, Hansen Life Sci. Research Building, Purdue University, West Lafayette, IN 47907, United States

SOURCE: Cancer Immunology, Immunotherapy, (2002) 51/3 (121-129).
Refs: 24
ISSN: 0340-7004 CODEN: CIIMDN

COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Monoclonal antibodies were generated in mice to a 34-kDa circulating form of a drug-responsive hydroquinone (**NADH**) **oxidase** with a protein disulfide-thiol interchange activity specific to the surface of cancer cells and the sera of cancer patients. **Screening** used Western blots with purified 34-kDa tNOX from HeLa cells and the sera of cancer patients. Epitopes were sought that inhibited the drug-responsive oxidation of NADH with the sera of cancer patients, but which had no effect on NADH oxidation with the sera of healthy volunteers. Two such antisera were generated. One, designated monoclonal antibody (mAb) 12.1, was characterized extensively. The **NADH oxidase** activity inhibited by mAb 12.1 also was inhibited by the quinone site inhibitor capsaicin (8-methyl-N-vanillyl-6-noneamide). The inhibition was competitive for the drug-responsive protein disulfide-thiol interchange activity assayed either by restoration of activity to scrambled RNase or by cleavage of a dithiodipyridine substrate, and was uncompetitive for NADH oxidation. Both the mAb 12.1 and the postimmune antisera immunoprecipitated drug-responsive NOX activity and identified the same 34-kDa tNOX protein in the sera of cancer patients that was absent from sera of healthy volunteers, and was utilized as immunogen. Preimmune sera from the same mouse as the postimmune antisera was without effect. Both mouse ascites containing mAb 12.1 and postimmune sera (but not preimmune sera) slowed the growth of human cancer cell lines in culture, but did not affect the growth of non-cancerous cell lines. Immunocytochemical and histochemical findings showed that mAb 12.1 reacted with the surface membranes of human carcinoma cells and tissues.

```
=>  d his

     (FILE 'HOME' ENTERED AT 12:56:53 ON 15 MAY 2002)

     FILE 'REGISTRY' ENTERED AT 12:56:58 ON 15 MAY 2002
                E AR-NOX/CN
                E NADH OXIDASE/CN
L1              1 S E3

     FILE 'CAPLUS, USPATFULL, BIOSIS, EMBASE, MEDLINE' ENTERED AT 12:57:44 ON
     15 MAY 2002
                E MORRE DOROTHY/AU
L2         105112 S E3 OR ER OR E5 OR E2
L3              7 S MORRE D/AU
                E MORRE D/AU
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L4          1756 S E3 OR E5 OR E6 OR E7
L5           304 S L4 AND L2
L6           186 DUP REM L5 (118 DUPLICATES REMOVED)
L7             0 S L6 AND AR-NOX
L8            59 S L6 AND NADH OXIDASE
L9        986566 S SCREEN OR SCREENING
L10            1 S L8 AND L9

=> s ubiquinone
L11        19869 UBIQUINONE

=> s cytochrome c or cyt c
L12       110680 CYTOCHROME C OR CYT C

=> s superoxide dismutase
L13       102002 SUPEROXIDE DISMUTASE

=> s ascorbate
L14        59299 ASCORBATE

=> s l8 and l11
L15            3 L8 AND L11

=> s l15 not l10
L16            3 L15 NOT L10

=> d ibib abs

L16  ANSWER 1 OF 3  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER:   1999217938  EMBASE
TITLE:              A multifunctional hydroquinone oxidase of the external
cell
                    surface and sera.
AUTHOR:             Morre D.J.; Pogue R.; Morre D.M.
CORPORATE SOURCE:   Prof. D.J. Morre, Med. Chem./Molec. Pharmacol. Dept.,
                    Purdue University, 1333 Hansen Life Sci. Res. Bldg., West
                    Lafayette, IN 47907-1333, United States
SOURCE:             BioFactors, (1999) 9/2-4 (179-187).
                    Refs: 27
                    ISSN: 0951-6433  CODEN: BIFAEU
COUNTRY:            Netherlands
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       029     Clinical Biochemistry
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   A multifunctional cell surface protein with NADH oxidase
     (NOX) activity and capable of oxidizing hydroquinones is located at the
     exterior of the cell and is shed in soluble form into sera. The oxidase
     appears to function as a terminal oxidase of a trans plasma membrane
     electron transport chain consisting of a NAD(P)H-ubiquinone
     reductase at the cytosolic membrane surface, possibly a b-type
cytochrome,
     ubiquinone and the oxidase. Hyperactivity or conditions that
     interrupt ordered 2H+ + 2e- transport from NAD(P)H or hydroquinone to
     molecular oxygen and other acceptors at the external cell surface may
     result in the generation of superoxide. The latter may serve to propagate
     aging-related redox changes both to adjacent cells and circulating blood
     components. A circulating NOX activity form associated with aging and the
     reduction of cytochrome c by sera of aged patients that is partially
     inhibited by ubiquinone are described.
```

=> d 2 ibib abs

L16 ANSWER 2 OF 3 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1999180341 EMBASE
TITLE: The plasma membrane **NADH oxidase** of HeLa cells has hydroquinone oxidase activity.
AUTHOR: Kishi T.; **Morre D.M.**; **Morre D.J.**
CORPORATE SOURCE: D.J. Morre, Department of Medicinal Chemistry, Purdue University, West Lafayette, IN 47907, United States. morre@pharmacy.purdue.edu
SOURCE: Biochimica et Biophysica Acta - Bioenergetics, (1999) 1412/1 (66-77).
Refs: 35
ISSN: 0005-2728 CODEN: BBBEB4
PUBLISHER IDENT.: S 0005-2728(99)00049-3
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical Biochemistry
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The plasma membrane **NADH oxidase** activity partially purified from the surface of HeLa cells exhibited hydroquinone oxidase activity. The preparations completely lacked NADH:**ubiquinone** reductase activity. However, in the absence of NADH, reduced coenzyme Q10 (Q10H2=ubiquinol) was oxidized at a rate of 15.+-.6 nmol min-1 mg protein-1 depending on degree of purification. The apparent K(m) for Q10H2 oxidation was 33 .mu.M. Activities were inhibited competitively by the cancer cell-specific **NADH oxidase** inhibitors, capsaicin and the antitumor sulfonylurea N-(4-methylphenylsulfonyl)-N'-(4-chlorophenyl)urea (LY181984). With coenzyme Q0, where the preparations were unable to carry out either NADH:quinone reduction or reduced quinone oxidation, quinol oxidation was observed with an equal mixture of the Q0 and Q0H2 forms. With the mixture, a rate of Q0H2 oxidation of 8-17 nmol min-1 mg protein-1 was observed with an apparent K(m) of 0.22 mM. The rate of Q10H2 oxidation was not stimulated by addition of equal amounts of Q10 and Q10H2. However, addition of Q0 to the Q10H2 did stimulate. The oxidation of Q10H2 proceeded with what appeared to be a two-electron transfer. The oxidation of Q0H2 may involve Q0, but the mechanism was not clear. The findings suggest the potential participation of the plasma membrane **NADH oxidase** as a terminal oxidase of plasma membrane electron transport from cytosolic NAD(P)H via naturally occurring hydroquinones to acceptors at the cell surface. Copyright (C) 1999 Elsevier Science B.V.

=> d 3 ibib abs

L16 ANSWER 3 OF 3 MEDLINE
ACCESSION NUMBER: 2000233862 MEDLINE
DOCUMENT NUMBER: 20233862 PubMed ID: 10769214
TITLE: Surface oxidase and oxidative stress propagation in aging.

AUTHOR: **Morre D M**; Lenaz G; **Morre D J**
CORPORATE SOURCE: Department of Foods and Nutrition, Purdue University, West Lafayette, IN 47907, USA.. morred@cfs.purdue.edu
SOURCE: JOURNAL OF EXPERIMENTAL BIOLOGY, (2000 May) 203 Pt 10 1513-21. Ref: 81
Journal code: I2F; 0243705. ISSN: 0022-0949.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000714
Last Updated on STN: 20000714
Entered Medline: 20000706

AB This report summarizes new evidence for a plasma-membrane-associated hydroquinone oxidase designated as CNOX (constitutive plasma membrane **NADH oxidase**) that functions as a terminal oxidase for a plasma membrane oxidoreductase (PMOR) electron transport chain to link the accumulation of lesions in mitochondrial DNA to cell-surface accumulations of reactive oxygen species. Previous considerations of plasma membrane redox changes during aging have lacked evidence for a specific terminal oxidase to catalyze a flow of electrons from cytosolic NADH to molecular oxygen (or to protein disulfides). Cells with functionally deficient mitochondria become characterized by an anaerobic metabolism. As a result, NADH accumulates from the glycolytic production of ATP. Elevated PMOR activity has been shown to be necessary to maintain the NAD(+)/NADH homeostasis essential for survival. Our findings demonstrate that the hyperactivity of the PMOR system results in an **NADH oxidase** (NOX) activity capable of generating reactive oxygen species at the cell surface. This would serve to propagate the aging cascade both to adjacent cells and to circulating blood components. The generation of superoxide by NOX forms associated with aging is inhibited by coenzyme Q and provides a rational basis for the anti-aging activity of circulating coenzyme Q.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	52.38	58.55

STN INTERNATIONAL LOGOFF AT 13:04:26 ON 15 MAY 2002